A CLINICAL LECTURE ON THE DIFFERENTIAL DIAGNOSIS OF ANTERO-LATERAL SCLEROSIS AND POSTERIOR SCLEROSIS OF THE SPINAL CORD.*

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THERE are two patients present to-day who exhibit very opposite manifestations of spinal disease, and yet they are very frequently confounded one with the other. I think, however, that when we come to examine them, we shall find that the symptoms are almost as different as those of any other two diseases with which the human body can become afflicted.

Before proceeding to examine those patients, I shall say a few words regarding the spinal cord, as the basis of the remarks I shall have to make. The spinal cord is not a simple organ. On the contrary, it is a compound organ, anatomically and physiologically. It has distinct anatomical features, and these, as a matter of course, have distinct physiological manifestations. As a consequence, we find that in spinal disease the symptoms exhibited bear an exact relation with the physiology of the part of the cord affected.

As you will see by the diagram, the spinal cord consists essentially of two masses of tissue. All that portion on the periphery is called the white substance, while that located centrally is called the gray substance.

The external portion is divided into various sections; but not to enter into details of the divisions, it will serve our purpose this afternoon to speak of this external portion as the antero-lateral and posterior columns.

The antero-lateral columns include all that part of the white matter on each side lying between the posterior horns of gray matter and the anterior median fissure. The pos-

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terior columns embrace that portion of each side lying between the posterior horns of gray matter and the posterior median fissure. This latter is divided into two parts, that lying nearer the median fissure being called the column of Goll, and that contiguous to the posterior horn of gray matter being called the column of Burdach. It is with this latter of the two portions of the spinal cord that we have to deal with in one of these cases, while the other, as I think I shall be able to show you, is affected with a disease of the antero-lateral column.

The posterior columns of the spinal cord have certain distinct functions; they relate to sensibility and co-ordination.

The column of Goll is primarily so seldom the seat of disease, and a post-mortem examination has been made on so few of the patients in whom it has been affected, that the diseased manifestations to which it gives rise are not definitely known. It has been pretty reasonably established, however, that the column of Goll is concerned, like the column of Burdach, with sensibility and to a certain extent with co-ordination. This column of Burdach, with which we are particularly concerned to-day, is sometimes called the posterior root zone, because the radicals which come from the posterior horns of gray matter seem to start from them. It is owing to pressure upon the radicals, due to the abnormal process characteristic to locomotor ataxia, that we have the sharp, shooting, lightning like pains which are almost invariably met with in cases of that disease.

It used to be supposed that the co-ordinating faculty resided in the cerebellum. Probably it does to some extent. The cerebellum certainly has something to do in maintaining the equilibrium of the body, and so have the semicircular canals of the auditory apparatus. But that function seems to be a little different from co-ordination. A person suffering from a disease of the cerebellum cannot, it is true, walk well, but his difficulty in walking is not due to his inability to co-ordinate well, but to vertigo.

You probably, in the course of your attendance upon physiological lectures, witnessed the removal of the cere-

bellum from pigeons; you have then noticed that the bird lodged upon the table unable to stand, and every attitude, every expression, seems to show that it suffers from vertigo; its eyes roll, and its head partakes of the same motion. When thrown into the air, it flies in a way which shows that it is subject to a peculiar sensation that causes it to act in a manner similar to that of a child that has turned around many times; it staggers in fact in the air, at the same time it does not appear to be deprived of the ability to co-ordinate its limbs when it desires to move them. That is, if you disturb its limbs individually, it will move them in a perfectly co-ordinate manner. I judge from these phenomena, and from the symptoms of patients suffering from cerebellar diseases, such as an abscess, a tumor, or an injury, that the difficulty in locomotion exhibited by them is due to vertigo. At any rate, in post-mortem examinations of persons who have suffered from locomotor ataxia the cerebellum is usually found in a state of health, while the column of Burdach in the spinal cord is found diseased.

The antero-lateral column has nothing whatever to do with sensibility, and of course a patient suffering from disease of this portion of the cord would exhibit no aberration whatever of sensibility, neither in paræsthesia nor anæsthesia, so long as the disease remained in that portion of the cord. Sometimes, however, the membranes of the cord become involved, and then there are some painful sensations. Again, the disease may spread to the posterior column, as it sometimes does, and then there is derangement of sensibility. But so long as the disease is confined to the antero-lateral column there is nothing whatever but derangement of motility, because there is nothing in that portion of the cord but motor fibres. That being the case, when you have patients affected with disease of the antero-lateral column you expect to find derangement of motion and nothing else. We will now see how these statements accord with the histories in these two cases. First, however, let us turn to one other point.

In sclerosis of the antero-lateral column and of the pos-

terior column the disease is always symmetrical. There are some diseases of the spinal cord which are not symmetrical. But in these two both sides are affected, and usually to the same extent. Not always exactly to the same extent, because it does not always affect both sides at the same time, and the disease gets ahead on one side faster than on the other. But after a year or two you will find the manifestations of the disease are about the same on one side as on the other.

With that basis for an examination of these patients we will take up their cases one at a time and see how they correspond with the facts just gone over.

The first patient tells us he is eighteen years of age; that he has been sick about four years. It dates from a fall on the back of the head. About four months afterward people began to notice that in his walk he kept next to the curbstone, or by the houses. After his attention had been called to this peculiarity in his walk, he found that unless he gave attention to where he stepped he would have a tendency to fall.

He says he did have pains in the legs, but they have disappeared. The pains were short and shooting. When asked whether he felt distinctly with the bottom of his feet he replies no, and when asked whether he seemed to be walking on cushions he replies that the bottom of his feet seemed to come to a point as if he were walking on skates, and they wabbled. He says he passes a great deal of water, but he is able to pass it when he desires. He cannot restrain it long after the desire comes. He has had feelings as if a rope were tied around the waist at about the crest of the ilium. He sometimes sees double, and things look blurred. He sometimes has pains in the head. He has had trouble with speech for sometime. There is no trouble with the arms; no numbness, he tells us; but on inquiry, we find that he is unable to pick up a pin until after several trials. He has trouble with his fingers; they do not feel natural.

The tongue is not tremulous. He says his food seems to stop when being swallowed. He complains of difficulty

in the pronunciation of words, and when asked to repeat the words "truly rural" and "national intelligencer," he does it with difficulty. He can repeat the sentence, "Peter Piper picked a peck of pickled peppers," but he does it very slowly and only by placing his whole attention upon it. I have given him these words to test his labials and linguals, —the power to use his lips and his tongue. Ordinarily we do not have to give our attention to speech which has become automatic; this young man, however, cannot pronounce linguals and labials without thought.

This patient has not the Argyle Robertson symptom of locomotor ataxia. In a patient having that symptom the pupil does not contract for light, but it does contract for accommodation. In this patient the pupil contracts for light and also for accommodation. His pupils are larger than usual in patients who have locomotor ataxia; in fact the pupils are usually much contracted in this disease, and are likely to be equal. This man's pupils are about equal; the right may be a little larger than the left, but they are both larger than we commonly observe in this disease.

None of the symptoms, as far as we have gone, are exactly characteristic of locomotor ataxia. A man might have all of his symptoms as far as we have discovered them, and yet have some other disease than locomotor ataxia. Now we will inquire with regard to some pathognomonic symptoms.

When asked to stand with his eyes closed, he separates his feet, widens his base; and when asked to walk with his eyes closed, he goes but a short distance when he begins to stagger and fall. He has lost his ability to bring his muscles into such harmonious action as will result in exact movement. He cannot place his feet where he wants to except by concentrating his whole attention upon his movements. If he were to try to mount a horse, he could not place his foot in the stirrup. If he were to try to get into a carriage, he would probably miss the step. He has lost to some extent the power of co-ordination, which is one of the symptoms of locomotor ataxia, and without which locomotor ataxia never exists.

But there are other diseases in which there is want of power of co-ordination besides locomotor ataxia. We see the same thing in multiple neuritis. That symptom is not, then, sufficient in itself to establish the existence of locomotor ataxia, although there cannot be locomotor ataxia without it.

We now proceed to test his patellar tendon reflex, and we find that it is present in nearly a normal degree. That settles the point beyond a doubt that this is not a case of locomotor ataxia pure and simple, for when that disease exists alone, there is always abolition of the knee-jerk; that is, when the legs are crossed and you strike just below the patellar, there is no rebound of the foot. I have never seen a case of pure locomotor ataxia in which there was exaggeration of the patellar tendon reflex. Not that the abolition of the tendon reflex is itself pathognomonic of the disease, for it is not. There are some people who never have had the tendon reflex, who have been born without it, who are not suffering with locomotor ataxia. But if you put those two symptoms together, the diminution or abolition of the patellar tendon reflex and co-ordination, such as is seen in this patient, they are sure indications that the person is suffering from locomotor ataxia, or sclerosis of the column of Burdock—the external part of the posterior portion of the spinal cord.

When I began the examination of this patient, I was as much surprised as any of you to find that the patellar tendon reflex had not been abolished, for I had been prepared to look for the further symptoms of locomotor ataxia which up to that point in the examination were present. But the case is proving all the more interesting, as it will probably enable me to demonstrate a combination of the two affections, the general anatomical features of which I have just described.

This patient speaks of a sensation of constriction around the waist. That line is supposed to mark the upper limit of the morbid process in the cord. But in many cases which have come under my observation I am perfectly satisfied that there has been disease above that line. Therefore I

am unable to explain satisfactorily what that sense of constriction is due to. I do not even know what the condition in the muscles or nerves is which gives rise to it. It may be a sort of contraction of the muscles, or it may be some trouble with the nerves themselves.

This patient states that his legs have not felt stiff, they do not shake, but they sometimes suddenly spring forward after a sleepy attack. He has not ankle clonus. The only regular symptom of locomotor ataxia which is absent in his case is the patella tendon reflex. This is not, I think, exaggerated, but it is not abolished. The only way in which we can account for that is the existence of a condition called spastic ataxia, which consists of a combination of locomotor ataxia and antero-lateral sclerosis.

It is characterized by the symptoms of both diseases to a certain extent. Suppose this man's symptoms began, as they probably did, in the posterior columns of the spinal cord, and that in the first place there was diminution or abolition of the tendon reflex. Suppose the morbid process extended until it involved the antero-lateral columns. The tendency of disease of the antero-lateral columns is to produce exaggerated tendon reflex. Consequently, as the disease advanced in this case, the loss of the tendon reflex would be replaced by its return or by an exaggeration of it. We cannot determine whether the disease began as an antero-lateral sclerosis or as a postero-lateral sclerosis until we shall have examined further. As long as the disease is confined to the posterior columns of the cord, there is no loss of power of motion. Let the patient straighten the leg, and you will find yourself unable to bend it at the knee. I have seen patients who could not stand with their eyes closed, nor walk with the eyes shut, nor feel the ground with the soles of their feet, yet they were as strong as you or I, and it was impossible to flex their legs against their will.

If this man has a combination of the two diseases under consideration, he has lost power in the legs. If he is as strong in the legs as he ever was, he has a disease which I have never seen. If he has the combination of locomotor

ataxia and antero-lateral sclerosis, he must necessarily have lost some strength in the legs, for in antero-lateral sclerosis there is loss of power.

For the purpose of comparison, we will now obtain the history of the second patient, who is supposed to have antero-lateral sclerosis. His age is forty-six. He says he has always been quite weak. By weakness he means his toes have always dragged on the ground; that is to say, he could not raise his foot. This is the manner in which antero-lateral sclerosis begins. He had a tendency to fall down. His legs got a little stiffer every year; they would jump at night, and they twitch occasionally yet. As you see, in his walk the toes drag on the ground. When asked to cross the legs while sitting, he is compelled to lift one leg over the other with the hands. When asked to lift the heel while the toes rest on the floor, he does so, and says that sometimes the foot begins to shake, but it rests quietly now because it is too stiff. When it starts going, he has no power whatever to stop it except by putting the heel down. He has, then, three symptoms which are characteristic of antero-lateral sclerosis, viz., rigidity, paralysis, and exaggerated tendon reflex.

If this patient should come back again in three or four years, we should find that he had no exaggerated tendon reflex as he now has, he would have lost that ankle clonus, but he would preserve his other symptoms and, in addition, he would have loss of co-ordinating power. The rigidity of the muscles is sometimes so great that when such patients in walking have contraction of the muscles on the anterior face of the thigh and legs, the one leg becomes locked over the other, and they are unable to proceed. This patient says he has not been troubled in that way.

When asked whether he has any pains, he replies that he has not, that he only feels stiff. Whatever pains he may have are due to stiffness in the muscles and to outside disturbances, not to disturbance in the cord.

When asked to stand with his feet close together and the eyes shut, he is able to do it without any unsteadiness whatever. The other patient, you will see, is quite unsteady.

The second patient is also able to walk as well with the eyes closed as with them open. The first patient says that in the beginning of his symptoms his legs felt too heavy, and that when sitting in a chair or lying on the sofa he imagined he was going to fall. That is not a characteristic symptom of locomotor ataxia. His legs, he says, never felt stiff, nor does he think they are weak. He imagines his legs would be all right if he could place his feet where he liked. On testing the strength of his legs, we find they can be bent at the knee very easily, while on one of the healthy men present we are unable to overcome the power of the extensors of the leg. That shows this patient must have some other affection than merely locomotor ataxia, for the strength of the muscles is not affected in the last-named disease. The explanation of this symptom would also account for the absence of the Argyle Robertson pupil, which is almost always seen.

There ought also to be something else, to which I have not yet alluded, which is a complicating feature. It is trouble with speech and swallowing. The patient says he sometimes chokes, that he has trouble in moving his tongue and in bringing his lips together. When saying "Peter Piper," etc., he did not bring his lips closely together. That is evidence that he has some bulbar disease in addition to antero-lateral and postero-lateral sclerosis; that is to say, he has indications of beginning glosso-labio-laryngeal paralysis. If he comes here within a year or two you will find that he cannot swallow, that the saliva dribbles from his mouth, that he has lost ability to articulate, and the lower part of his face is almost, if not quite, paralyzed. All this, he says, came from the blow on the back of his head. That would account for the bulbar disease, but it would not in itself account for the other symptoms—those depending on disease of the spinal cord. But it might all be accounted for by the commencement of the disease lower down in the cord, and ascending, involving the other parts. He now says that his eyesight is blurred. All the symptoms go to show advancing disease, having already reached the ganglia at the base of the brain, affecting the third pair of nerves and the optic nerves.

When we began to examine this patient we expected to find locomotor ataxia. While I am disappointed, I am glad that it turned out not to be a simple case of that disease. Besides the interest in the case itself, it illustrates very well the importance of not stopping and making a diagnosis after elucidating what appear to be one or two characteristic symptoms of any affection which you think the patient may have. I was astonished to see his leg fly out when testing the patellar tendon reflex. It was something which I had not at all expected, for the other symptoms pointed to uncomplicated locomotor ataxia. But that occurrence disabused me of my first impression of the nature of his disease. And so you will often find that as you proceed with the examination of a case you may change your mind several times as to its real nature. All that is instructive.

The third patient before you is a woman, past her thirtieth year, who, when questioned regarding the symptoms we have discovered in these other patients, seems to think that she too has some of them. She makes believe that there is ankle clonis at both ankles, and so with some other symptoms. Instead of having antero-lateral sclerosis, she is simply hysterical, and has no affection of the cord at all. There is no real disease about her, at least none such as we are looking for. All that is simply simulation of disease. Her walk shows that she has no paralysis, no locomotor ataxia; the manner in which she stands shows that she co-ordinates well. The symptoms which she has are only those which she has seen us experimenting with and which she has imitated, although but poorly. She is hysterical; she has a disease, a disorder, but it is not such as these other patients have. I had her come in simply to show one of the manifestations of hysteria—how it may simulate anything at all. I had a woman patient four or five years ago who was strongly hysterical, and who of all persons whom I have seen was most under the influence of the principle of suggestion. I could make her believe anything at all. I suppose we can generally do that with women, but at the same time we cannot always impose on them in the way I could impose on that one. I would take

up a book, for instance, ask "What is that?" She would reply, "It is a book." I would say, "It is not a book, it is a watch." She would say, "No, it does not look like a watch." I would say, "Look at it again, and see if it does not look like a watch." She would then say, "It does look like a watch." "Have you the hardihood to tell me it is not a watch?" "I beg your pardon, doctor; it is a watch." "What sort of a watch?" "I hardly know." "Is it a silver watch?" "Yes." "Now look at it again, and you will see it is not silver; it is gold." "Oh, yes, doctor; it is gold."

With such persons you could do anything, make them believe anything, for they lose the power of voluntarily directing their thought. The patient before us is one of that kind, except that she has more volition remaining than had the one to whom I have referred.

Let us return for a few minutes to the first two cases.

In the one, we have found that he has an affection of two portions of the cord; that he has locomotor ataxia and also antero-lateral sclerosis. Consequently his gait is not purely that of locomotor ataxia, it is a mixture of the gait peculiar to these two affections. The gait of locomotor ataxia is perfectly characteristic, and I am enabled by it to tell while patients are walking through a passage way of some thirty feet to enter my office that they have locomotor ataxia, although I may never have seen them. Such patients make two distinct movements with the foot and two distinct sounds. This patient does sometimes, but not always. If he had pure locomotor ataxia he would do it always. The heel strikes first, making a distinct sound, and then the sole strikes, making a second sound.

This patient has not very well defined symptoms in the sole of his feet, such as patients suffering from locomotor ataxia usually have. They usually consist of sensations as if the patient were walking on a cushion, on sand, or velvet, or as if the feet were too large for the boots, or as if something were crowding the toes. At the same time this patient has some change in sensation in the sole of the feet. He describes it as if walking on skates, and as if his feet were inclined to roll. He probably exhibits in a very mild degree,

if at all, the retardation of sensibility which is one of the characteristic symptoms of locomotor ataxia. By that I mean that the impression made on the cutaneous surface of the feet, for instance, is not appreciated by the brain for a space of time considerably longer than would be necessary in a person normally constituted. Sometimes that interval is so long that it is noticed by every person who may be in the room. I had a patient once at my clinic at the Bellevue Hospital Medical College suffering from locomotor ataxia in whom, as he walked about, I stuck two pins, one into the calf of each leg, up to the head. Some persons present took out their watches, and it was found that two minutes and a half elapsed before he felt the pins, and he then jerked up the feet as if something hurt him. As he said, you might pour boiling water on his legs and he would not know it until the flesh had fallen off.

I do not believe this patient is affected in that way. I do not suppose it will take more than a fraction of a second in his case for the sensation produced by the stick of a pin into the feet to travel to the brain. You see he complains apparently immediately after he is pricked, and there seems to be no diminution of sensibility. It is about the same on both sides.

Sensibility travels along the nerves at about the rate of eighty-one feet a second. Call it over four feet to his brain, he ought to feel the sensation in about one-twentieth of a second, and that probably is about the time in which he does feel it. You can appreciate, then, how great the retardation must have been in the patient at Bellevue College.

In the second patient, who has antero-lateral sclerosis, we do not expect any retardation of sensation, for he has no trouble with the posterior columns of the cord; and he has no retardation of sensation as is shown by experiment.

Another word with regard to the reason why the first patient is unable to stand with the eyes closed. He can stand much better with the eyes open, and he can walk pretty well when he has use of his sight. We know that when walking in the street we do not have to look where

we are placing our feet, or just the place where we are going. We look and get a general idea of the direction, but we do not watch our feet, nor do we have any guide-post before us. But these patients cannot walk in that way. They must have a guiding spot somewhere. As long as a patient suffering from locomotor ataxia keeps his eyes on the guiding spot he walks pretty well. As the disease advances he shortens that spot, brings it nearer, until finally he has to keep his eyes directed to the floor and see where he is next to place his feet. The moment he finds his eyes off of his feet he begins to fall. That shows that there is something in us when well, aside from the eyesight, which enables us to walk. That is called by some persons the muscular sense. We know, in a way which we do not understand, the exact state of contraction that our muscles are undergoing. If you take off all your clothing and bend your arm, you will know just the degree to which you are bending it without looking at it. How do you know that your arm is bending, or how much it is bent? I do not know. But it is attributed to the muscular sense. These patients do not know what use they are making of their muscles by that sense. They have lost the muscular sense, and cannot tell what their muscles have done unless assisted by sight. They have to see what they are doing. Whether that muscular sense can be called a sense or not, there is no doubt that it is a perception of some kind which conveys the impression to the brain and gives cognizance of what is going on. This patient has lost that perception not only in the legs, but also to some extent in the arms. When he is asked to close his eyes and bring the index finger of the outstretched hand to the tip of the nose or to the middle of the eyebrow, he misses the mark by half an inch, and he does not fully correct the mistake after several trials. Those of us who are in normal health make very slight, if any, error, even at the first trial, and when we miss the mark at the first trial, we correct it at the second.

One reason why I brought these two cases before you to-day was to sharply define the difference between the symptoms of locomotor ataxia and antero-lateral sclerosis

of the cord. The case illustrating the former disease turned out to be not a simple, but a complicated case, and therefore somewhat confusing. Almost every case of antero-lateral sclerosis which is sent to me by physicians who have not made more or less a specialty of diseases of the nervous system is sent as one of locomotor ataxia. You now see how essentially distinct are the two affections. The distinction is not only a matter of scientific import, it is also one which directly concerns the patient, as the treatment is different in the two classes of cases. Antero-lateral sclerosis is much more amenable to treatment than is locomotor ataxia. Of all the organic affections of the spinal cord, locomotor ataxia is the most common, while it is the least frequently cured. We shall have to postpone the consideration of the treatment until a future date.